IN THE SPECIFICATION:

Please amend paragraph numbers **[0093] – [0096], [0100], [0108], [0156],** and **[0172]** as follows. Please replace paragraphs **[0187]** and **[0188]** with replacement paragraphs **[0187]** and **[0188]**. No new matter is being added. Replacement paragraphs **[0187]** and **[0188]** are to correct typographical errors in Tables 4 and 5.

[0093] FIG. 5. Cytotoxic T cell activity against peptide pulsed and mHag positive target cells by two ex vivo induced HA-1 (a, b) and two ex vivo induced HA-2 specific CTLs (c, d). CTLs shown in a,c,d are induced using PBDC, whereas CTLs shown in b are induced using BMDC. Target cells: autologous PHA blasts (◊~~.diamond.~~); autologous PHA blasts pulsed with peptide (♦?); EBV-LCL positive for HA-1 (n=4) or HA-2(n=3) (Δ?); EBV-LCL negative for HA-1 (n=3) or HA-2(n=3) (○?); HA-1 or HA-2 negative EBV-LCL pulsed with HA-1 or HA-2 peptide (●~~.endot.~~).

[0094] FIG. 6. Hematopoietic cell restricted cytolysis mediated by in vivo (a,c) and ex vivo (b,d) induced HA-1 (a,b) and HA-2 (c,d) specific CTLs. All target cells were derived from the same HLA-A2+, HA-1+, HA-2+blood donor. Target cells: PHA blasts (Δ?); fibroblasts (♦?); Fibroblasts cultured with IFN-γ?+ TNF-a (both 300 U/ml) (●~~.endot.~~); Fibroblasts cultured with IFN-γ? plus TNF-αa and pulsed with 10 μ~~.mu.~~g/ml peptide (○?).

[0095] FIG. 7. Lysis of HA-1.sup.+ (a,b,c) or HA-2.sup.+ (d,e,f) positive leukemic cells by in vivo (b,e) and ex vivo (c,f) induced HA-1 and HA-2 specific CTLs. Lysis of target cells by control HLA-A2 specific CTL clone is shown in a and d. Target cells: HA-1 or HA-2 negative EBV-LCL (_), HA-1 or HA-2 positive EBV-LCL (Δ?), Leukemic cells positive for HA-1 (n=4) or HA-2 (n=3) (♦?), HA-1 or HA-2 positive leukemic cells cultured with IFN-γ?+ TNF-αa (●~~.endot.~~).

[0096] FIG. 8. HA-1 gene expression in hematopoietic and non-hematopoietic cells. The relative HA-1 gene expression levels were determined by a calibration function generated from RNA of

the HA-1 positive KG-1 cell line. Cells of hematopoietic origin tested were: *PBMCs (n=3), Dendritic cells (n=6), + Langerhans cells (n=2), ~~.English Pound.~~ EBV-LCLs (n=5), uPHA blasts (n=6), ~~.sup.-~~ Mast cell lines (n=3), Monocytes (n=4), Thymocytes (n=Cells of non-hematopoietic origin tested were: Keratinocytes (n=5), ~~.cent.~~ Fibroblasts (n=2), - PTECs (n=3), r HUVECs (n=3), s Melanocytes (n=3), two SV40 immortalized breast cell lines: ~~.sup.-~~ HaCat and HBL-100.

[0100] FIG. 12. CGH profile of cell PN3-C1. Each chromosome is represented by its ideogram and numbered. ~~Deletions are marked with a red bar (e.g. loss of chr.13) at the left and gains with a green bar (e.g. gain of chr. 8q) at the right side of the chromosome symbol.~~

[0108] Cytotoxic T cell clones specific for the mH antigen HA-1 have been isolated from three different patients with severe GvHD.sup.7. The mH antigen HA-1 is presented in the context of HLA-A2.1 and present in 69% of the HLA-A2.1 positive population.sup.7. HA-1 expression was demonstrated to be tissue specific and limited to cells of hematopoietic origin, including dendritic cells, Langerhans cells and leukemic cells.sup.8-10. Family analysis indicated a Mendelian mode of inheritance for HA-1 and segregation independent from the MHC complex.sup.11. Comparison of the T cell receptor (TCR) sequences of different HA-1 specific T cell clones derived from different individuals revealed conserved usage of the TCR Vβ~~b~~6.9 and conserved amino acids in the CDR3 region.sup.12. In a retrospective study, mismatching for a number of mH antigens was evaluated with regard to the association with GvHD after HLA-identical BMT. A single HA-1 mismatch between donor and recipient was significantly correlated with the induction of GvHD after HLA-identical BMT.sup.3.

[0156] The polymorphic HA-1.sup.H and HA-1.sup.R regions were screened with the HLA-peptide binding prediction software of BIMAS (Bioinformatics & Molecular Analysis Section, NIH, Bethesda, Md.; url: bimas.dcrt.nih.gov ~~http://bimas.dcrt.nih.gov./~~) for octameric, nonameric or decameric HA-1 peptides capable to bind to HLA class I molecules. The selection of peptide candidates was made by comparison of the computed scores with that of the HLA-A2 restricted HA-1.sup.H CTL epitope with amino acid (aa) sequence VLHDDLLEA (SEQ ID NO:2)

(score=79.6). This score corresponds to the estimated half-time of dissociation of complexes containing the peptide at 37~~.degree.~~ °C~~.~~ at pH 6.5. Five HA-1.sup.H/R peptides with scores ranging from 32 (intermediate binding score) to 176 (strong binding score) were selected to assay for binding to the relevant HLA class I molecules. The predicted HLA class I/HA-1.sup.H/R peptide associations and their computed binding scores are presented in table 3. In addition, we selected two decameric HA-1.sup.H/R peptides that contained anchor residues for binding to HLA-A3 but were not predicted by the BIMAS software. With reference to Table 3, peptide number 1 is represented by SEQ ID NO: 34, peptide number 2 is represented by SEQ ID NO: 35, peptide number 3 is represented by SEQ ID NO: 36, peptide number 4 is represented by SEQ ID NO: 37, peptide number 5 is represented by SEQ ID NO: 38, peptide number 6 is represented by SEQ ID NO: 39, peptide number 7 is represented by SEQ ID NO: 4, peptide number 8 is represented by SEQ ID NO: 5, peptide number 9 is represented by SEQ ID NO: 6, peptide number 10 is represented by SEQ ID NO: 7, peptide number 11 is represented by SEQ ID NO: 40, peptide number 12 is represented by SEQ ID NO: 23, peptide number 13 is represented by SEQ ID NO: 41, peptide number 14 is represented by SEQ ID NO: 42, peptide number 15 is represented by SEQ ID NO: 2, peptide number 16 is represented by SEQ ID NO: 10, peptide number 17 is represented by SEQ ID NO: 43, peptide number 18 is represented by SEQ ID NO: 44, peptide number 19 is represented by SEQ ID NO: 45, peptide number 20 is represented by SEQ ID NO: 46, peptide number 21 is represented by SEQ ID NO: 47, and peptide number 22 is represented by SEQ ID NO: 48.

[0172] Twenty-nine amino acid long HA-1.sup.H/R peptides were subjected to in vitro digestion with EBV-LCL derived 20S immuno-proteasomes. Within a time frame of 15 minutes, major peptide fragments were cleaved at the COOH-termini of both nonameric and decameric HLA-B60 binding HA-1.sup.H/R peptides. The latter cleavage products contained the intact HLA-B60 binding sequences with 3-5 additional amino acid residues at the N termini for the HA-1.sup.H and HA-1.sup.R peptides as demonstrated in Table 4 and Table 5, respectively. Thus, both the HA-1.sup.H and the HA-1.sup.R products can be effectively cleaved by proteasomes to generate the precursors of the peptides that bind to HLA-B60. A 29 amino acid long HA-1^{A} peptide is represented by SEQ ID NO: 78. Fragments 1 through 9 in Table 4 are represented by SEQ ID

NOS: 79-87, respectively. A 29 amino acid long HA-1^R peptide is represented by SEQ ID NO: 88. Fragments 1 through 13 of Table 5 are represented by SEQ ID NOS: 89-101.

[0187]

TABLE 4

In vitro proteasomal cleavage of a 29 amino acid long HA-1^A peptide

% fragment digested in			G L E K L K E C V L H D D L L E A R R P R A H E C L G E A
15 min	30 min	45 min	
17.2	22.4	0	G L E K L K E C V L H D D L
14.7	11.8	14.7	G L E K L K E C V L H D D L L E A R R P R A H E C L G
13.9	16.4	21.7	H D D L L E A R R P R A H E C L G E A
13.0	10.3	13.9	G L E K L K E C V L H D D L L E A R R P R A
10.5	8.3	12.1	E K L K E C V L H D D L L
9.6	8.8	12.3	G L E K L K E C V L H D D L L E A R R P R A H E C
8.5	9.2	13.8	G L E K L K E C V L H D
7.8	7.4	11.5	G L E K L K E C V L H D D L L E A
4.8	5.5	0	G L E K L K E C V L

The peptide sequences that bind to HLA-B60 are underlined.

The amounts of the generated fragments after cleavage with 20s imniuno proteasomes for 15, 30 and 45 min are expressed an the percentage of all fragments found in the digested substrate.

[0188]

TABLE 5

In vitro proteasomal cleavage of a 29 amino acidlong HA-1^R peptide

% fragment digested in			G L E K L K E C V L R D D L L E A R R P R A H E C L G E A
15 min	30 min	45 min	
26.2	28.0	23.8	G L E K L K E C V L R D D L L E A R R P R A H E C L G
14.0	16.0	13.5	G L E K L K E C V L R D D L L E A R R P R A H E C L G E
11.1	14.3	12.7	G L E K L K E C V L R D D L
7.9	9.6	7.9	G L E K L K E C V L R D D L L E A R R P R A
6.6	8.6	8.3	E K L K E C V L R D D L L
6.2	7.5	7.3	C V L R D D L L E A R R
5.3	7.0	5.7	G L E K L K E C V L R D D L L E A R R P R A H E C L
4.9	7.1	6.4	G L E K L K E C V L R D D L L E A R R P R A H E C
4.2	6.1	5.6	G L E K L K E C V L R D
3.9	4.2	3.9	G L E K L K E C V L R D D L L E A
3.6	4.4	4.4	G L E K L K E C V L R D D L L E A R R
3.4	4.2	3.6	G L E K L K E C V L R D D L L E A R R P R
2.6	4.2	4.0	G L E K L K E C V L R D D L L E A R R P R A H

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The amounts of the generated fragments after cleavage with 20s immuno proteasomes for 15, 30 and 45 min are expressed as the percentage of all fragments found in the digested substrate.